



Meigs Syndrome Masquerading as Advanced Ovarian Malignancy

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Case Report

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Abstract

Meigs syndrome — the classic triad of benign ovarian tumour, ascites, and pleural effusion — is a well-recognised yet diagnostically treacherous mimic of advanced ovarian malignancy. We report a 34-year-old nulliparous woman who presented with a 30–32-week-size solid right adnexal mass, bilateral pleural effusions necessitating intercostal drainage, haemodynamic compromise, and a markedly elevated serum CA-125 of 629 U/mL. Colour Doppler sonography showed absent internal vascularity, and computed tomography of the chest revealed bilateral effusions with mediastinal shift. Following multidisciplinary stabilisation, staging laparotomy identified a 20×20 cm right ovarian mass with a twisted pedicle but no peritoneal deposits. Intraoperative frozen section confirmed a benign ovarian stromal tumour (fibrothecoma), enabling fertility-preserving right salpingo-oophorectomy. The postoperative course was uneventful; effusions resolved and CA-125 trended toward normal. This case underscores how completely Meigs syndrome can masquerade as advanced ovarian malignancy, and demonstrates that a grossly elevated CA-125 alone does not exclude a benign diagnosis. Intraoperative frozen section can be helpful in guiding fertility-preserving surgery in this young woman.

Keywords: Meigssyndrome; Ovarian; Fibrothecoma; CA-125; Pleural Effusion; Frozen Section; Fertility-Preserving Surgery

Introduction

Meigs syndrome, first described by Joe Vincent Meigs and John W. Cass in 1937, is defined by the triad of a benign solid ovarian tumour — most commonly a fibroma or fibrothecoma — accompanied by ascites and ipsilateral or bilateral pleural effusion, all of which resolve completely following surgical excision of the tumour [1]. Despite being a well-characterised entity, its preoperative distinction from Stage III–IV epithelial ovarian carcinoma remains one of the most formidable diagnostic challenges in gynaecological practice.

The diagnostic difficulty arises because the clinical, biochemical, and radiological profile of Meigs syndrome can be virtually indistinguishable from advanced ovarian malignancy. Both conditions may present with a large solid

pelvic mass, gross ascites, pleural effusions with respiratory compromise, and strikingly elevated serum CA-125 — an epithelial tumour marker with well-documented non-specific elevation in benign conditions including endometriosis, pelvic inflammatory disease, hepatic cirrhosis, and Meigs syndrome itself [2,3].

Ovarian fibromas account for approximately 4% of all ovarian tumours, yet Meigs syndrome complicates only 1–3% of these [4]. The rarity of the condition, paradoxically, makes it a more dangerous diagnostic pitfall: clinicians who encounter it infrequently may not readily include it in the differential diagnosis of a young woman with a massive solid adnexal mass, florid ascites, and bilateral pleural effusions. The consequence of misdiagnosis — unnecessary radical cytoreductive surgery — carries serious implications for fertility and quality of life, particularly in young nulliparous

women. We present this case to highlight the clinical, biochemical, and intraoperative features that should alert the clinician to the possibility of Meigs syndrome, and to highlight the potential role of intraoperative frozen section in enabling conservative, fertility-preserving surgery.

Case Report

A 34-year-old nulliparous woman was referred to the Department of Obstetrics & Gynaecology, Christian Medical College & Hospital, Ludhiana, with a four-month history of progressive abdominal distension, increasing breathlessness on minimal exertion, bilateral lower limb swelling, and anorexia. She had no prior gynaecological history of note, no family history of ovarian or breast malignancy, no prior surgeries, and was not receiving any hormonal therapy.

On general examination, the patient was in respiratory distress with oxygen saturation of 89% on room air, pulse rate 120/min, and blood pressure 90/60 mmHg. There was bilateral pitting oedema of the lower limbs. Respiratory examination revealed bilateral decreased air entry. Abdominal examination demonstrated a large, firm, non-tender, mobile pelvic-abdominal mass of approximately 30–32 weeks gestational size with restricted mobility, mild tenderness, and associated massive ascites.

Serum CA-125 was markedly elevated at 629 U/mL (reference < 35 U/mL). Haemogram showed Hb 12.1 g/dL, TLC 38,300/ μ L, and platelets 4,90,000/ μ L. Other tumour markers (CEA, AFP, beta-hCG, LDH) were within normal limits. Renal and liver function tests were acceptable. Ultrasound of the abdomen demonstrated a large heterogeneous solid right adnexal mass measuring approximately 18.5×19 cm with absent internal vascularity on colour Doppler and associated right hydronephrosis. NCCT chest confirmed bilateral pleural effusions (left > right) with consolidation-collapse of the underlying lung parenchyma and mediastinal shift (Figure 1). CT abdomen and pelvis showed a large right adnexal solid mass, massive ascites, no omental thickening, no peritoneal deposits, and normal left adnexa.

The patient was managed with multidisciplinary input from gynaecological oncology, respiratory medicine, and anaesthesiology. Bilateral intercostal drainage (ICD) was inserted, yielding large volumes of straw-coloured fluid; cytology was negative for malignant cells. Following haemodynamic resuscitation, nutritional optimisation, and respiratory stabilisation over 72 hours, staging laparotomy was performed under general anaesthesia.

Intraoperatively, approximately 200 mL of residual ascitic fluid was aspirated. A 20×20 cm solid right ovarian mass weighing 1,200 g, with a twisted pedicle and smooth,

bosselated surface, was identified. There were no peritoneal deposits, omental involvement, or lymphadenopathy. The uterus and left adnexa were grossly normal. Intraoperative frozen section of the ovarian mass reported interlacing spindle-cell bundles, bland elongated nuclei, and collagenous stroma consistent with a benign ovarian fibroma/fibrothecoma (Figure 1). On this histological confirmation, a fertility-preserving right salpingo-oophorectomy was performed, conserving the uterus and left adnexa.

The postoperative period was uneventful. ICDs were removed sequentially on days 2 and 3 as effusions resolved. The patient was discharged on postoperative day 6 in satisfactory condition. Post-treatment chest radiograph confirmed complete bilateral effusion resolution (Figure 1-8). Final histopathology confirmed benign ovarian fibrothecoma with areas of haemorrhagic necrosis, moderate acute-on-chronic inflammatory infiltrate, no atypia, and no mitotic activity. Chronic salpingitis of the right fallopian tube was also noted. At six-week follow-up, the patient was asymptomatic and CA-125 had declined to 38 U/mL.



Figure 1: Cxr Showing Bilateral Pleural Effusions with Mediastinal Shift (Pre-Treatment).

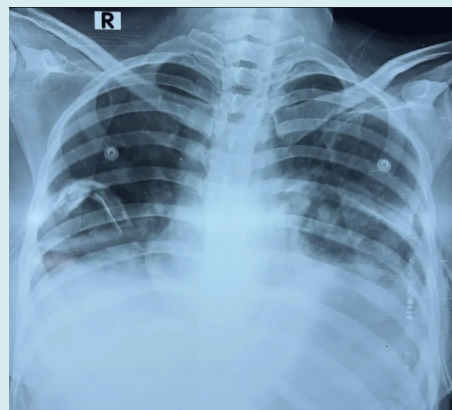


Figure 2: Cxr Confirming Bilateral Effusion Resolution After Icd Drainage (Post-Treatment).

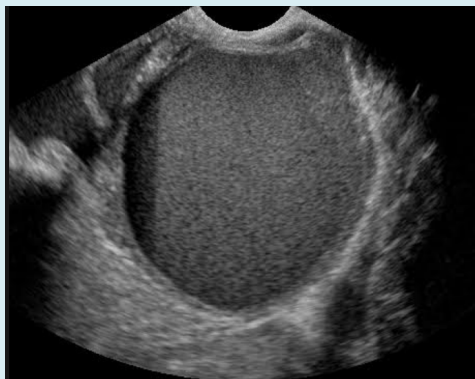


Figure 3: Ultrasound Demonstrating The 18.5×19 Cm Solid Right Adnexal Mass.



Figure 4: NCCT Abdomen Coronal View Showing the Large Right Adnexal Solid Mass (Blue Arrows).

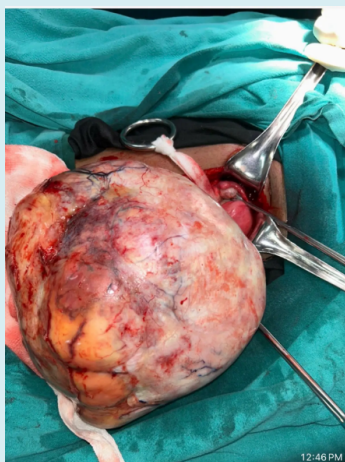


Figure 5: Intraoperative View of the 20×20 Cm Right Ovarian Mass with Twisted Pedicle through Midline Laparotomy Incision.

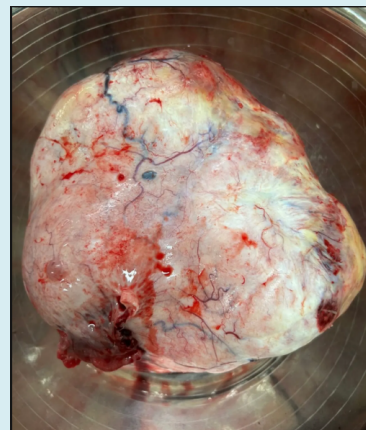


Figure 6: Gross Specimen Post Right Salpingo-Oophorectomy Showing Smooth, Firm White-Grey Capsule with Right Tube.

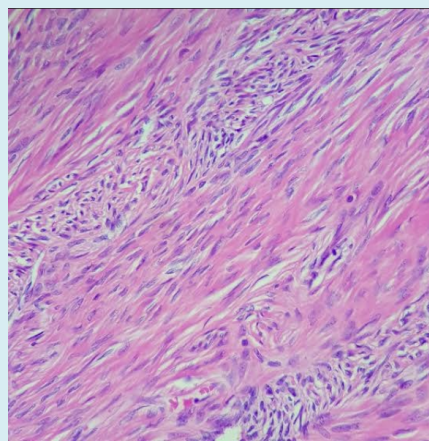


Figure 7: Frozen Section: Interlacing Spindle-Cell Bundles with Bland Elongated Nuclei and Collagenous Stroma Consistent with Ovarian Fibroma.

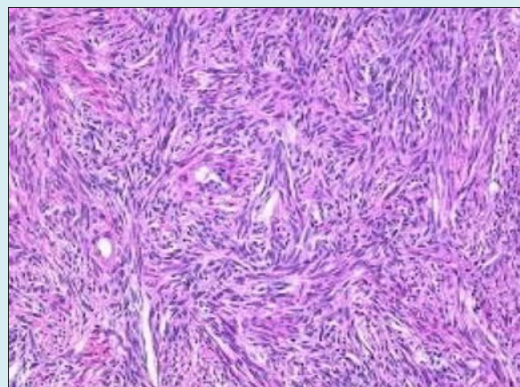


Figure 8: H&E ×100 Confirming Ovarian Fibroma/Thecoma on Final Histopathology.

Discussion

This case exemplifies the classical diagnostic trap of Meigs syndrome presenting as presumed advanced ovarian malignancy. The combination of a massive solid adnexal mass, bilateral pleural effusions, respiratory compromise, gross ascites, and CA-125 of 629 U/mL would, in the majority of clinical encounters, generate an overwhelming working diagnosis of Stage III–IV epithelial ovarian carcinoma. Yet the diagnosis was ultimately benign, confirming that Meigs syndrome must always be considered in the differential diagnosis, particularly in young women.

Elevated CA-125 in Meigs Syndrome. CA-125 elevation in Meigs syndrome is well documented. The prevailing mechanism relates to mesothelial irritation: ascites and pleural effusion cause upregulation of CA-125 expression from peritoneal and pleural mesothelial surfaces, independent of malignancy [2,5]. Reported CA-125 levels in Meigs syndrome range from mildly elevated values to those exceeding 1000 U/mL [3,6]. Vascular endothelial growth factor (VEGF), which increases capillary permeability, has also been implicated in fluid accumulation, with VEGF levels shown to decline significantly after tumour removal [5,7].

Imaging Features. Colour Doppler remains important in pre-operative adnexal mass evaluation. Absent internal vascularity, as in our case, is a feature more consistent with fibrous or stromal tumours than with vascular malignant neoplasia [8]. CT findings of a smooth capsule, massive ascites without omental thickening or peritoneal deposits, and normal contralateral adnexa further supported a benign aetiology. In ambiguous cases, MRI with diffusion-weighted imaging provides superior tissue characterisation [8].

Pedicle Torsion. An additional intraoperative finding was a twisted pedicle — a recognised but infrequently reported complication of large ovarian fibromas [9]. Torsion likely contributed to the haemodynamic compromise seen on admission. Large tumour size and mobility are established predisposing factors.

Pathophysiology of Effusions: The dominant hypothesis for pleural effusion in Meigs syndrome involves transdiaphragmatic migration of ascitic fluid through diaphragmatic lymphatics, typically producing right-sided effusion. Bilateral effusions, as in this patient, represent a more severe manifestation associated with larger tumours and greater ascitic volumes [10]. Complete resolution after tumour removal is the diagnostic hallmark.

Role of Frozen Section: Intraoperative frozen section was the pivotal intervention. Confirmation of benign fibrothecoma enabled right salpingo-oophorectomy rather than radical

cytoreductive surgery, preserving this nulliparous patient's uterus and contralateral adnexa [11]. Current evidence supports frozen section as having adequate sensitivity and specificity for intraoperative assessment of stromal ovarian tumours [11].

Differential Diagnosis: The differential includes pseudo-Meigs syndrome, advanced epithelial ovarian carcinoma, peritoneal mesothelioma, tuberculous peritonitis, and cardiac or hepatic causes of transudative effusions [12]. Cytological analysis of ascitic fluid — a transudate negative for malignant cells in Meigs syndrome, as in our case — is a further diagnostic tool [13-18].

Conclusion

Meigs syndrome can present with clinical, biochemical, and radiological features virtually indistinguishable from advanced ovarian malignancy. A markedly elevated serum CA-125 alone should not preclude consideration of this benign diagnosis. Absent internal vascularity on Doppler and absent peritoneal deposits on CT are valuable pre-operative clues. Intraoperative frozen section can be helpful: it provides real-time histological confirmation and can be the key decision point enabling conservative, fertility-preserving surgery. Timely recognition avoids unnecessary radical procedures and preserves reproductive potential.

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Conflict of Interest

The authors declare no conflict of interest, financial or otherwise.

Patient Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying clinical images.

Author Contributions

Awalroop Kular: Concept, clinical care, manuscript drafting, and revision. Vaibhav Bhatti: Data collection and literature search. Prof. Kavita Mandrelle: Supervision, critical revision, and final approval. All authors have read and approved the final manuscript.

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